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 NEWS  2              "Ask CAS" for self-help around the clock
 NEWS  3  SEP 09      CA/CAplus records now contain indexing from 1907 to the
                      present
 NEWS  4  DEC 08      INPADOC: Legal Status data reloaded
 NEWS  5  SEP 29      DISSABS now available on STN
 NEWS  6  OCT 10      PCTFULL: Two new display fields added
 NEWS  7  OCT 21      BIOSIS file reloaded and enhanced
 NEWS  8  OCT 28      BIOSIS file segment of TOXCENTER reloaded and enhanced
 NEWS  9  NOV 24      MSDS-CCOHS file reloaded
 NEWS 10  DEC 08      CABA reloaded with left truncation
 NEWS 11  DEC 08      IMS file names changed
 NEWS 12  DEC 09      Experimental property data collected by CAS now available
                      in REGISTRY
 NEWS 13  DEC 09      STN Entry Date available for display in REGISTRY and CA/CAplus
 NEWS 14  DEC 17      DGENE: Two new display fields added
 NEWS 15  DEC 18      BIOTECHNO no longer updated
 NEWS 16  DEC 19      CROPU no longer updated; subscriber discount no longer
                      available
 NEWS 17  DEC 22      Additional INPI reactions and pre-1907 documents added to CAS
                      databases
 NEWS 18  DEC 22      IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
 NEWS 19  DEC 22      ABI-INFORM now available on STN
 NEWS 20  JAN 27      Source of Registration (SR) information in REGISTRY updated
                      and searchable
 NEWS 21  JAN 27      A new search aid, the Company Name Thesaurus, available in
                      CA/CAplus
 NEWS 22  FEB 05      German (DE) application and patent publication number format
                      changes

 NEWS EXPRESS  DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
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FILE 'HOME' ENTERED AT 09:13:07 ON 11 FEB 2004

=> file medline biosis embase caplus
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
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=> s ouabain (p) voltage (p) sensitive (p) dye (action (p) potential
MISSING OPERATOR 'DYE (ACTION'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s ouabain (p) voltage (p) sensitive (p) dye (p) action (p) potential
L1             4 OUABAIN (P) VOLTAGE (P) SENSITIVE (P) DYE (P) ACTION (P) POTENTI
                 AL

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2             1 DUP REM L1 (3 DUPLICATES REMOVED)

=> d l2 ibib kwic

L2   ANSWER 1 OF 1     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:    97308260     MEDLINE
DOCUMENT NUMBER:     97308260   PubMed ID: 9165486
TITLE:               Membrane depolarization in LA-N-1 cells. The effect of
                     maitotoxin is Ca(2+)- and Na(+)-dependent.
AUTHOR:              Sorrentino G; Monsurro M R; Singh I N; Kanfer J N
CORPORATE SOURCE:    Institute of Neurological Sciences, Faculty of Medicine,
                     2nd University of Naples, Italy.
SOURCE:              MOLECULAR AND CHEMICAL NEUROPATHOLOGY, (1997 Apr) 30 (3)
                     199-211.
                     Journal code: 8910358. ISSN: 1044-7393.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199707
ENTRY DATE:          Entered STN: 19970805
                     Last Updated on STN: 19970805
                     Entered Medline: 19970718
AB   We investigated the influence of ion compositions on the membrane
     potential in LA-N-1 human neuroblastoma cells using bisoxonol as a
     potential-sensitive fluorescent dye. The
     ability of K+, ouabain, veratridine, and maitotoxin to induce
     membrane depolarization was evaluated. Increasing concentrations of K+
     ions from 10 to 50 mM caused a dose-dependent increase of bisoxonol
     fluorescence, which was completely independent on Na+ and Ca2+.
     Ouabain (5 mM), an inhibitor of the Na+, K(+)-ATPase, failed to
     induce membrane depolarization. Veratridine (40 and 100 microM), a Na+
     channel activator, only in the presence of 10 micrograms of Leiurus
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scorpion venom reduced the membrane **potential.** Maitotoxin (MTX) from 3 to 10 ng/mL depolarized LA-N-1 cells in a dose-dependent manner, and produced a rapid and sustained. . . Na+ ions also seem to be, although only partially, implicated in the MTX effects, since both the blockade of tetrodotoxin (TTX)-**sensitive voltage** -operated Na+ channels and the removal of Na+ ions were able to reduce the depolarization. In conclusion, our data indicate that the depolarizing **action** of MTX on LA-N-1 cells is Ca(2+)- and Na(+)-dependent, although the latter only partially, and that this effect is dependent on Ca2+ influx into the cells likely through a **voltage**-insensitive calcium-entry system.

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=> d his

     (FILE 'HOME' ENTERED AT 09:13:07 ON 11 FEB 2004)

     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 09:13:25 ON 11 FEB 2004
L1             4 S OUABAIN (P) VOLTAGE (P) SENSITIVE (P) DYE (P) ACTION (P) POTE
L2             1 DUP REM L1 (3 DUPLICATES REMOVED)

=> s voltage (p) sensitive (p) dye (p) action (p) potential (p) sodium (p) channel
(p) block?
L3            10 VOLTAGE (P) SENSITIVE (P) DYE (P) ACTION (P) POTENTIAL (P) SODIU
                 M (P) CHANNEL (P) BLOCK?

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4             5 DUP REM L3 (5 DUPLICATES REMOVED)

=> d l4 total ibib kwic

L4   ANSWER 1 OF 5     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:    96419791     MEDLINE
DOCUMENT NUMBER:     96419791   PubMed ID: 8822549
TITLE:               Propagation of action potentials in the dendrites of
                     neurons from rat spinal cord slice cultures.
AUTHOR:              Larkum M E; Rioult M G; Luscher H R
CORPORATE SOURCE:    Department of Physiology, University of Bern, Switzerland.
SOURCE:              JOURNAL OF NEUROPHYSIOLOGY, (1996 Jan) 75 (1) 154-70.
                     Journal code: 0375404. ISSN: 0022-3077.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         199611
ENTRY DATE:          Entered STN: 19961219
                     Last Updated on STN: 19961219
                     Entered Medline: 19961112
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AB 1. We examined the propagation of **action potentials** in the dendrites of ventrally located presumed motoneurons of organotypic rat spinal cord cultures. Simultaneous patch electrode recordings were made from the dendrites and somata of individual cells. In other experiments we visualized the membrane **voltage** over all the proximal dendrites simultaneously using a **voltage-sensitive dye** and an array of photodiodes. Calcium imaging was used to measure the dendritic rise in Ca2+ accompanying the propagating **action potentials.** 2. Spontaneous and evoked **action potentials** were recorded using high-resistance patch electrodes with separations of 30-423 microm between the somatic and dendritic electrodes. 3. **Action potentials** recorded in the dendrites varied considerably in amplitude but were larger than would be expected if the dendrites were to. . . passive cables (sometimes little or no decrement was seen for distances of > 100 microm). Because the amplitude of the **action**

potentials in different dendrites was not a simple function of distance from the soma, we suggest that the conductance responsible for the boosting of the **action potential** amplitude varied in density from dendrite to dendrite and possibly along each dendrite. 4. The dendritic **action potentials** were usually smaller and broader and arrived later at the dendritic electrode than at the somatic electrode irrespective of whether. . . occurred at the dendrite or soma or as a result of spontaneous synaptic activity. This is clear evidence that the **action potential** is initiated at or near the soma and spreads out into the dendrites. The conduction velocity of the propagating **action potential** was estimated to be 0.5 m/s. 5. The **voltage** time courses of previously recorded **action potentials** were generated at the soma using **voltage** clamp before and after applying 1 microM tetrodotoxin (TTX) over the soma and dendrites. TTX reduced the amplitude of the **action potential** at the dendritic electrode to a value in the range expected for dendrites that behave as passive cables. This indicates that the conductance responsible for the actively propagating **action potentials** is a Na+ conductance. 6. The amplitude of the dendritic **action potential** could also be initially reduced more than the somatic **action potential** using 1-10 mM QX-314 (an intracellular **sodium channel blocker**) in the dendritic electrode as the drug diffused from the dendritic electrode toward the soma. Furthermore, in some cases the **action potential** elicited by current injection into the dendrite had two components. The first component was **blocked** by QX-314 in the first few seconds of the diffusion of the **blocker.** 7. In some cells, an afterdepolarizing **potential** (ADP) was more prominent in the dendrite than in the soma. This ADP could be reversibly **blocked** by 1 mM Ni2+ or by perfusion of a nominally Ca2+-free solution over the soma and dendrites. This suggests that the back-propagating **action potential** caused an influx of Ca2+ predominantly in the dendrites. 8. With the use of a **voltage-sensitive dye** (di-8-ANEPPS) and an array of photodiodes, the **action potential** was tracked along all the proximal dendrites simultaneously. The results confirmed that the **action potential** propagated actively, in contrast to similarly measured hyperpolarizing pulses that spread passively. There were also indications that the **action potential** was not uniformly propagated in all the dendrites, suggesting the possibility that the distribution of Na+ **channels** over the dendritic membrane is not uniform. 9. Calcium imaging with the Ca2+ fluorescent indicator Fluo-3 showed a larger percentage change in fluorescence in the dendrites than in the soma. Both bursts and single **action potentials** elicited sharp rises in fluorescence in the proximal dendrites, suggesting that the back-propagating **action potential** causes a concomitant rise in intracellular calcium concentration...

L4 ANSWER 2 OF 5 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 92333334 MEDLINE
DOCUMENT NUMBER: 92333334 PubMed ID: 1378490
TITLE: Maitotoxin-induced intracellular calcium rise in PC12 cells: involvement of dihydropyridine-sensitive and omega-conotoxin-sensitive calcium channels and phosphoinositide breakdown.
AUTHOR: Meucci O; Grimaldi M; Scorziello A; Govoni S; Bergamaschi S; Yasumoto T; Schettini G
CORPORATE SOURCE: Department of Human Communicative Sciences, II School of Medicine, University of Naples, Italy.
SOURCE: JOURNAL OF NEUROCHEMISTRY, (1992 Aug) 59 (2) 679-88. Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199208
ENTRY DATE: Entered STN: 19920904
Last Updated on STN: 20021218
Entered Medline: 19920820

AB . . . calcium concentration and are always associated with an increase of the free cytosolic calcium level. We tested the effects of **voltage-sensitive** calcium **channel blockers** (nicardipine and omega-conotoxin) on maitotoxin-induced intracellular calcium increase, membrane depolarization, and inositol phosphate production in PC12 cells. Maitotoxin dose dependently. . . fluorescent probe fura 2. This effect disappeared in a calcium-free medium; it was still observed in the absence of extracellular **sodium** and was enhanced by the dihydropyridine calcium agonist Bay K 8644. Nicardipine inhibited the effect of maitotoxin on intracellular calcium. . . was reduced by pertussis toxin pretreatment. Maitotoxin caused a substantial membrane depolarization of PC12 cells as assessed by the fluorescent **dye** bisoxonol. This effect was reduced by pretreating the cells with either nicardipine or omega-conotoxin and was almost completely abolished by. . . in a calcium-free EGTA-containing medium. The findings on maitotoxin-induced cytosolic calcium rise and membrane depolarization suggest that maitotoxin exerts its **action** primarily through the activation of **voltage-sensitive** calcium **channels**, the increase of inositol phosphate production likely being an effect dependent on calcium influx. The ability of nicardipine and omega-conotoxin to inhibit the effect of maitotoxin on both calcium homeostasis and membrane **potential** suggests that L- and N-type calcium **channel** activation is responsible for the influx of calcium following exposure to maitotoxin, and not that a depolarization of unknown nature causes the opening of calcium **channels.**

L4 ANSWER 3 OF 5 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 91109787 MEDLINE
DOCUMENT NUMBER: 91109787 PubMed ID: 2177149
TITLE: Bretylium causes a K(+)-Na+ pump activation that is independent of Na+/H+ exchange in depolarized rat, mouse and human lymphocytes.
AUTHOR: Tron L; Pieri C; Marian T; Balkay L; Emri M; Damjanovich S
CORPORATE SOURCE: Biomedical Cyclotron Laboratory, University Medical School of Debrecen, Hungary.
SOURCE: MOLECULAR IMMUNOLOGY, (1990 Dec) 27 (12) 1307-11.
Journal code: 7905289. ISSN: 0161-5890.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199102
ENTRY DATE: Entered STN: 19910329
Last Updated on STN: 19910329
Entered Medline: 19910226

AB We have studied a bretylium tosylate induced increase of the membrane **potentials** of partially depolarized rat, mouse and human lymphocytes, using the **potential sensitive dye** , bis [1,3, dibutylbarbituric acid-(5) trimethine oxonol]. The extent of this repolarization is dose-dependent and decreased in magnitude as the temp was reduced from 37 degrees C to room temperature The repolarizing effect is inhibited by K(+)-Na(+)-pump **blockers** or lack of extracellular Na+. **Sodium** ion **channel blockers** are effective in abolishing repolarization only if applied prior to, or simultaneously with, bretylium. Activation of Na+/H+ exchange is not. . . is completely eliminated in the presence of 10 microM amiloride (concn of the diuretics having no measurable inhibition on the **action** of the exchanger). These data suggest that bretylium opens ligand- and **voltage**-gated Na+ **channels**

, and repolarization occurs due to higher activity of the K(+)-Na(+)-pump stimulated by the enhanced intracellular Na+ accumulation.

L4 ANSWER 4 OF 5 MEDLINE on STN
ACCESSION NUMBER: 89138590 MEDLINE
DOCUMENT NUMBER: 89138590 PubMed ID: 2852172
TITLE: Optical recording of electrical activity from axons and glia of frog optic nerve: potentiometric dye responses and morphometrics.
AUTHOR: Konnerth A; Orkand P M; Orkand R K
CORPORATE SOURCE: Max-Planck-Institute of Biophysical Chemistry, Gottingen-Nikolausberg, Federal Republic of Germany.
CONTRACT NUMBER: NS 07464 (NINDS)
NS-24913 (NINDS)
SOURCE: GLIA, (1988) 1 (3) 225-32.
Journal code: 8806785. ISSN: 0894-1491.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198904
ENTRY DATE: Entered STN: 19900306
Last Updated on STN: 19970203
Entered Medline: 19890406

AB **Voltage-sensitive dyes** were used to study the changes in membrane **potential** in axons and glial cells of the frog optic nerve following electrical stimulation. The lack of a signal in the unstained nerve and the multiphasic **action** spectra after staining indicated that the optical responses were from the extrinsic **dyes.** Changes in **dye** absorption and fluorescence had rapid and slow phases. The rapid phases resulted from **action potentials** in myelinated and unmyelinated axons. The kinetics of the slow phase of the optical response were similar to the depolarization. . . . with intracellular electrodes. The ratio of the amplitudes of the fast and slow phases was characteristic for each type of **dye.** Pharmacological analysis of the **action potential** of the unmyelinated axons revealed tetrodotoxin-**sensitive sodium channels** and 4-aminopyridine-**sensitive** potassium **channels.** Repeated exposure of the stained preparation to light led to photodynamic damage as shown by a **block** of recovery of the glial depolarization. An electron microscopic morphometric study of the nerve was carried out in an effort. . . . membrane was much greater than was the ratio of the fast and slow components of the signal, suggesting that the **dyes** either had a higher affinity for glial membrane or did not penetrate the nerve uniformly.

L4 ANSWER 5 OF 5 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 80026362 EMBASE
DOCUMENT NUMBER: 1980026362
TITLE: The effects of some organic 'calcium antagonists' on calcium influx in presynaptic nerve terminals.
AUTHOR: Nachshen D.A.; Blaustein M.P.
CORPORATE SOURCE: Dept. Physiol. Biophys., Washington Univ. Med. Sch., St Louis, Mo. 63110, United States
SOURCE: Molecular Pharmacology, (1979) 16/2 (579-586).
CODEN: MOPMA3
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
002 Physiology
008 Neurology and Neurosurgery
LANGUAGE: English

AB The **actions** of the organic 'Ca antagonists' verapamil and D-600 were tested on pinched-off presynaptic nerve terminals (synaptosomes) from rat brain, and. . . was measured in control media, and in depolarizing media containing either 75 mM potassium or veratridine, an alkaloid that opens **sodium channels.** The extra uptake induced by depolarizing media appears to be mediated by **voltage-sensitive** Ca **channels.** Synaptosome depolarization was indirectly determined with the **voltage-sensitive** fluorescent **dye**, di-pentyl oxacarbocyanine. Verapamil or D-600 (100 μM) inhibited the K+-induced 45Ca uptake by about two thirds, but had no effect on the K+-induced synaptosome depolarization; this inhibition of Ca uptake is, presumably, due to **block** of Ca-**channels.** Veratridine-induced 45Ca influx was more than 80% inhibited by verapamil or D-600 (100 μM), and veratridine-induced depolarization was almost completely **blocked.** These observations indicate that Na **channels** as well as Ca **channels** are inhibited by verapamil and D-600. Recordings of miniature end-plate **potentials** were used to evaluate the **actions** of verapamil and D-600 at the frog neuromuscular junction, after miniature end-plate **potential** frequency had been made **sensitive** to changes in the bathing Ca concentration by raising the external K+. Miniature end-plate **potential** frequency was not affected by verapamil (40-50 μM) or D-600 (10 μM) but was significantly reduced by Mn2+ (0.2 mM), a known **blocker** of Ca **channels.** Although verapamil and D-600 appear to be very potent antagonists of Ca currents in heart and smooth muscle, we conclude that Ca **channels** in vertebrate neurons are much less **sensitive** to these drugs.

=> log y

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FULL ESTIMATED COST	33.62	33.83

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